

AperTO - Archivio Istituzionale Open Access dell'Università di Torino

Nanosponge-cyclodextrins functionalized with oxygen protects H9C2 cells from hypoxia/reoxygenation injury: Implications from an in vitro model

This is the author's manuscript

Original Citation:

Availability:

This version is available <http://hdl.handle.net/2318/1699720> since 2019-04-23T12:35:59Z

Published version:

DOI:10.1016/j.vph.2017.12.021

Terms of use:

Open Access

Anyone can freely access the full text of works made available as "Open Access". Works made available under a Creative Commons license can be used according to the terms and conditions of said license. Use of all other works requires consent of the right holder (author or publisher) if not exempted from copyright protection by the applicable law.

(Article begins on next page)

Nanosponge-cyclodextrins functionalized with oxygen protects H9C2 cells from hypoxia/reoxygenation injury: Implications from an in vitro model

S. Femminò , F. Bessone , F. Caldera , R. Cavalli , F. Trotta , P. Pagliaro , C. Penna

Department of Clinical and Biological Sciences, University of Turin, Italy; Department of Chemistry, University of Turin, Italy; Department of Drug Science and Technology, University of Turin, Italy

Objective: Nanoparticle-based imaging and nanocarriers therapies have emerged as essential tools for many fields of modern medicine, in order to track the fate of cells and optimize drug delivery. Up to now, however, there are only few reports on the effect of nanocarriers of different types on oxygen delivery, even though this would be of great interest for the design of high impact therapies in several cardiovascular diseases (CVDs). In particular, Cyclodextrin Nanosponges (C-NS) can be envisioned as innovative tools to improve the delivery of oxygen in a controlled manner in CVDs.

Methods: We tested oxygenated C-NS (OX-C-NS) at different concentrations (0.2, 2 and 20 µg/ml) for their capability to reduce cell mortality during hypoxia and reoxygenation (H/R) protocols. For comparative purpose, we also tested “blank materials” (C-NS filled with nitrogen gas without oxygen) and the effects of C-NS in Normoxia. To test the effectiveness of C-NS, we used H9c2, a cardiomyoblast cell line derived from rat heart, exposed to Normoxia (5% CO₂ and 21% O₂) or Hypoxia (5% CO₂ and 95% N₂) in a Hypoxic Chamber. The cellular mortality was measured with MTT assay.

Results: In Normoxia, regardless of OX-C-NS formulation, the H9c2 cells displayed a tendency to an increased proliferation, which seemed somewhat correlated to the concentration of OX-C-NS used. The different concentration of OX-C-NS, applied before Hypoxia, induced a significant reduction of cell mortality compared to C-NS without oxygen. Also, the application of OX-C-NS at the beginning of reoxygenation induced a marked reduction of cell death.

Conclusions: OX-C-NS may induce H9c2 cell proliferation in Normoxia and may protect H9c2 from H/R injury in vitro. The administration of oxygen in a controlled manner during or after an ischemic event may be an innovative approach for reduction of Ischemia/Reperfusion injury, with consequent reduction of chronic CVDs. Our preliminary results, and in particular the observation of a remarkable efficacy in reoxygenation, suggest an interesting potentiality for medical application of C-NS during the treatment of myocardial infarction. Further studies are required to ascertain the protective potential of C-NS on cardiac I/R injury under in vivo conditions.

doi:10.1016/j.vph.2017.12.021